bined antiplatelet therapy (ASS and clopidogrel) for the next 3 months.

Discussion

In the literature, associations of the PTA with intracranial aneurysms, cavernous sinus fistulas and trigeminal neuralgia were described [6–8]. Consistent with a previously published case report [9], our study emphasizes that the PTA may also play a role in cerebral ischemia while transmitting an embolus from the carotid artery distribution into the posterior circulation. In most instances, angiograms of patients with the PTA show a flow direction of the anastomosis from the carotid to the basilar artery [1]. This explains why patients with carotid stenoses proximal to the PTA may develop embolic infarcts in the territory of the posterior circulation. In our patient, a moderate stenosis of the right ICA progressed to a high-grade, hemodynamically relevant stenosis, and our hypothesis is that a former orthograde perfusion of the anastomosis turned to a retrograde flow contributing to the collateral supply of the right anterior circulation. After successful endovascular recanalization of the ICA stenosis, the orthograde flow of the PTA was reestablished and visible on the carotid angiogram.

Conclusions

In patients with otherwise unexplained upper brain stem ischemia and 'coincidental' stenosis of the ICA, further diagnostic efforts, preferably MRI and MR angiography [10], are justified to search for a persisting PTA. This may help to avoid potentially harmful delays in initiating specific treatment.

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Neuroimaging and Pathology of the Progression of Unilateral to Bilateral Medial Medullary Stroke

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Medial medullary stroke (MMS) accounts for less than 0.5% of all cerebral infarcts [1]. Spiller in 1908 [2] and Déjerine in 1914 [3] described the association of ipsilateral tongue paralysis and contralateral hemiparesis with loss of deep sensation, but without pathologic support.

This triad is rare and partial syndromes are more common [4]. Some autopsies have revealed bilateral involvement [4]. Ocular bobbing and upbeat nystagmus have also been reported [4].

We present the first case of a patient with initial presentation of right anteromedial medullary stroke and progression to bilateral anteromedial medullary ischemia documented by brain magnetic resonance imaging (MRI) as well as autopsy.

Case Presentation

A 64-year-old African-American woman presented with left hemiparesis sparing the face and dysphonia. She had a history of hypertension, hepatitis C, and chronic renal insufficiency. On examination she had upbeat nystagmus, labile blood pressure, tachycardia and right tongue deviation. Her deep tendon reflexes were brisk on the left side, tone was decreased but extensor plantar response was not present until few days later.

Initial head CT was normal but diagnosis of medullary infarct was entertained and confirmed by brain MRI. Diffusion-weighted imaging (DWI) revealed right medial medullary infarct (fig. 1a). Magnetic resonance angiography showed absent right vertebral artery flow and tortuous basilar artery (fig. 1b). She was started on intravenous heparin since presentation. One day later, she developed right hemiparesis and respiratory distress requiring intubation. Repeated DWI revealed bilateral MMS (fig. 2). Transesophageal echocardiogram, carotid and transcranial Doppler were negative. Hepatitis C screening and ANA were positive (1:80, mixed speckled and homogeneous pattern) and ESR was 74. Rheumatoid factor and antiphospholipid panel were negative. Patient had no history of drug abuse and initial drug screen was negative.

She remained in a locked-in state with marked fluctuation of the nystagmus and dysautonomia. Although initially she was able to communicate by blinking, few days later she became unable to move her tongue or facial muscles. She developed medical complications with worsening renal failure requiring dialysis. Two months later she developed unexplained fever. She had a gastric perforation caused by a PEG tube. She was treated for peritonitis but died a few days later. Autopsy revealed cystic bilateral medial medullary infarct with right vertebral artery stenosis and 50% basilar artery stenosis (fig. 3). Both vertebral arteries were patent but no flow was observed in the right posterior inferior cerebellar artery.

Discussion

This is the first documented brain MRI series showing the progression of unilateral anteromedial to bilateral anteromedial med-

Fig. 1. **a** DWI of the lower medulla, which demonstrates the initial right MMS. **b** Lack of flow in the right vertebral artery. Tortuous basilar artery can also be seen.

Fig. 2. DWI of the lower medulla demonstrates progression of the initial right medullary infarction to bilateral anteromedullary involvement.

Fig. 3. Cross-section specimen of the lower medulla from the autopsy, which demonstrates significant cystic lesion on the right medulla and softening of the left medial medulla.

ullary strokes. A previous report documented the progression of lateral medullary to total hemimedullary syndrome [5], but to our knowledge no report described the pattern we observed in our patient.

Previous case series reported a favorable outcome after MMS [6]. However, our patient had an unfortunate course, resulting in

death due to peritonitis and sepsis. Interestingly her first sign of peritonitis was unexplained fever. She lacked the characteristic signs of peritoneal irritation, which can occur in patients with high spinal cord or brainstem injury.

Autopsy confirmed the neuroimaging findings and showed cystic bilateral MMS with partial recanalization of the vertebral arter-

ies and 50% basilar artery stenosis confirming the thrombotic nature of the stroke. No cardiac source of embolism was found. Therefore, given the initial evidence of right vertebral artery occlusion by magnetic resonance angiography, we suspect that the progression to bilateral involvement was due to occlusion of a penetrating branch, although unfortunately the autopsy report did not focus on the origins of the anterior spinal arteries and no cerebral angiogram was performed.

MMS accounted for less than 0.5% of all cerebral infarcts in one series [1] and a similar number was reported in a 700-autopsy report [7]. Up to 1996, only 40 well-documented cases were reported [6], and this condition still remains a diagnostic challenge because of the heterogeneous clinical presentations [6].

In our patient the presence of upbeat nystagmus was an important diagnostic clue. It is thought to be secondary to medial longitudinal fasciculus involvement, although others proposed the involvement of the nucleus intercalatus of Staderini [8]. However, previous reports underemphasized the diagnostic importance of the upbeat nystagmus [1], including the largest case series of MMS published in 1995 and 2004 [9, 10] because of the heterogeneous clinical presentation of the MMS.

In summary, we present the first concomitant DWI and autopsy-documented case of progression of right to bilateral anteromedial medullary ischemia. It also points to the need of early suspicion of peritonitis or other abdominal infection in patients with bilateral brainstem ischemia and unexplained fever. Similar to patients with spinal cord injury, they may develop threatening abdominal infections without the classical signs of peritoneal irritation [11].

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Cerebral Hemodynamics and Autoregulation in Reversible Posterior Leukoencephalopathy Syndrome Caused by Pre-/Eclampsia

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Introduction

Eclampsia accounts for nearly 50% of ischemic strokes during pregnancy and puerperium [1]. ‘Pre-eclampsia’ is defined as hypertension with proteinuria, edema or both induced by pregnancy. Neurological manifestations range from diffuse vegetative signs to focal deficits like amaurosis and sensorimotor impairment. In case of additional seizures or coma, the condition is called ‘eclampsia’. Pre-/eclampsia is thought to arise from immunological changes during trophoblast invasion, leading to chronic placental ischemia and release of vasoactive cytokines and mediators.

The underlying cerebrovascular pathophysiology is still poorly understood. On computed tomography and magnetic resonance imaging (MRI), pre-/eclampsia presents with the typical morphological lesion pattern of ‘reversible posterior leukoencephalopathy syndrome’ (RPLS) which is characterized by a multifocal, often symmetric, vasogenic brain edema, predominantly in the posterior portions of the cerebral white matter and cortex [2].

During severe pre-/eclampsia, transcranial Doppler sonography (TCD) showed increased blood flow velocities in the basal cerebral arteries [3–7]. It is unclear whether this indicates cerebral hyperperfusion, vasospasm or both. Single-photon emission computed tomography revealed focal hyperperfusion [8], while angiographic studies reported intracranial vasospasms [9, 10].

Although cerebral dysautoregulation is thought to be of major influence on the development of RPLS and eclamptic encephalopathy, it has rarely been confirmed by autoregulation testing. We thus studied cerebral hemodynamics and autoregulation in 4 consecutive patients with pre-/eclampsia.

Case Descriptions

Patients and Methods. Between August 2001 and April 2003, four healthy primiparous women with normal pregnancy and puerperium until onset of pre-/eclampsia were admitted to our intensive-care unit. Severe pre-/eclampsia was defined as diastolic blood pressure ≥ 90 mm Hg or systolic blood pressure ≥ 140 mm Hg with proteinuria >0.3 g/l in a 24-hour collection, edema or both. As a control group for assessment of cerebral autoregulation, 8 age-matched healthy nonpregnant women and 5 healthy pregnant women (week of gestation 34–37) were studied. Extracranial duplex sonography was carried out using standard equipment (HDI 3500, ATL, USA). TCD measurements were performed with a Multi Dop $\times 4$ using a 2-MHz probe (DWL, Sipplingen, Germany) in all basal cerebral arteries. The resistance index (RI) was calculated from the waveform of cerebral blood flow velocity (CBFV) by the formula $RI = (CBFV_{systolic} - CBFV_{diastolic}) / CBFV_{systolic}$. Dynamic